Fish Poisoning in the Eastern Caribbean

ROBERT W. BRODY
Caribbean Research Institute
College of the Virgin Islands
St. Thomas, U.S. Virgin Islands 00801

INTRODUCTION

"When I first got here I thought that I'd be able to practically exist on fish. . . . ."; "I was poisoned 15 times before I left my father's house: he always liked to buy the big fish because there was more meat. . . . ."; "I can't get a contract for grouper or snapper with any of the hotels: they buy the same fish from Santo Domingo for 10 cents a pound more. . . . ."; "Where are all the seafood restaurants? I thought this was an island. . . . ." These comments and others like them represent a biotoxicological problem which has always plagued the eastern Caribbean. Ciguatera fish poisoning, scombroid poisoning and to a lesser extent "clupeoid", tetraodontoid and elasmobranch poisoning have been reported since pre-Columbian time and the problem shows no sign of lessening.

This paper will present the basic facts of fish poisoning in the eastern Caribbean area as we presently know them. It will not attempt to review the voluminous literature from the Pacific; the reader is referred to excellent summaries of Halstead (1967) and Banner (1971). At the present time we have very little "hard data" on the chemistry, biogenesis and biology and pharmacology of the fish poisoning problem in the area. We have yet to confirm that the most important type of poisoning, ciguatera, is in fact identical to the toxin from the Pacific. The sections on ciguatera are therefore based on our local observations and inferences from studies in the Pacific are noted.

Fish poisoning in the eastern Caribbean can be broken down into three major groupings. The endotoxins from the puffer-like fishes with the additional rarely reported cases of clupeoid, elasmobranch and hallucinogenic fish poisoning form the first group. These incidents represent a very small percentage of the total number of cases reported; several years of casual data-gathering and 6 months of active research have only producted two accounts in the last 4 years, both of clupeiotoxism.

The biology, chemistry and pharmacology of puffer fish poisoning have been accurately summarized elsewhere (Bagnis, 1970). Although the world-wide fatality rate is high (61%), very few cases of tetraodon poisoning are reported in the Caribbean, probably because the puffers are not highly esteemed as a food fish. This may also be the reason for the low incidence of reports of elasmobranch poisoning in the area. Shark has been harvested recently in an effort to produce a packaged seafood product ("Sea Flake") with generally good customer acceptance; there have been no instances of elasmobranch poisoning brought to our attention as a result. Hallucinatory fish poisoning (icthyoallyeinotoxism) is also an apparently rare event in the eastern Caribbean; it has been described to us by an herbalist on St. Thomas but an actual case history has not been received.

Clupeoid poisoning is a form of icthyosarcotoxism caused by ingestion of the flesh of fishes of the order Clupeiformes. The symptoms are frequently violent with an extremely high case fatality rate. The onset of symptoms is noted with a sharp, metallic taste in the mouth followed rapidly by severe gastrointestinal upset with distinct indications of vascular collapse (drop in blood pressure, cyanosis). This may be accompanied or rapidly followed by neurological disturbances (nervousness, dilated pupils, violent headache, tingling) and in severe cases respiratory distress, convulsions, coma and death. The toxin is apparently particularly virulent: death may occur within 15 minutes. The literature reports that persons have died while in the act of eating the fish — "part of the fish was still in the victim's mouth at the time of death" (Halstead, 1967).

Fish in the families Clupeidae (herrings), Engraulidae (anchovies), Albulidae (bonefishes) and Elopidae (tarpons) have been reported as toxic in the Caribbean; our experience has been limited to two cases involving the clupeoid fishes *Harengula humeralis* (yellow-billed sprat) and *Opisthonema oglinum* (thread herring). One case is reported from a fisherman on Tortola, B.V.I. who (with his family of five) ate "yellow bill sprat" (probably *Opisthonema oglinum* from his description of a "spine on the back fin") caught in Great Harbor, Peter Island, B.V.I. He reported "bad fish poisoning" symptoms generally similar to ciguatera poisoning; the rapid onset reported above was present. He forced all of the members of his family to vomit and all took a "heavy dose of sulphur" (a patent medicine). He did not contact local public health authorities. The family recovered from acute symptoms within 36 hours. (This incident took place in late 1967 and was reported in April 1971; the interview was thus clouded by time and at least two subsequent incidents of ciguatera poisoning.) The second case is reported by Halstead (1970) from Antigua, W.I. from 1968 (?) when "some small surface-feeding 'herring-like' fish were eaten." Two people died in this outbreak. Halstead suggests that local terminology of "yellow-billed sprat" is applicable to *Harengula humeralis.* The violence of the episode suggests that this case was an example of classic clupeiotoxism while the Tortola case is not definitely separable from ciguatoxism.

The chemical nature and biogenesis of clupeiotoxism is not presently known. Numerous authors have suggested that fish caught during the summer months are more likely to be toxic. All reported incidents are from fish caught close to tropical islands. There are several references in the literature to planktonic blooms as the causative organism, specifically a "monad" (dinoflagellate?) (Halstead, 1967); *Skujaella* (=*Trichodesmium*) (Randall, 1958) or to "swarming of palolo worms" (Halstead, 1967). It is probable that the toxin is produced by some planktonic form as the clupeoid fishes are for the most part plankton feeders; this may add strength to the position that clupeiotoxism is a separate entity from ciguatera poisoning. Clupeiotoxin is not thought to be degraded by normal cooking and the degree of freshness does not seem to have effect on the toxicity.

Clupeiotoxism may pose a wider threat to public health than the occasional locally consumed fish. If the toxin is indeed caused by "blooming" plankton organisms the likelihood of a large school of toxic fish cannot be overlooked. The sardine and anchovy groups are frequently thought of as ideal fish for Fish Protein Concentrate production; we have no data on the ability of current FPC technology to eliminate the toxin from the raw fish.

The second major group of poisonings experienced in the eastern Caribbean is the result of bacterial decomposition of fresh fish. In the Virgin Islands and throughout the northern Leeward Islands fish are typically sold "fresh" from the boat. Very few fishermen use ice or gut the fish before sale, in fact there is a strong feeling among older citizens that such preservation is used to camouflage the true "freshness". Eastern Caribbean fishermen do not use live-wells and fish frequently spend a good part of the day in the sun. These conditions obviously tend to promote bacterial decomposition of the fish and the consequent toxicity problems; it is interesting that the younger people in St. Thomas, U.S. V.I. who tend to buy fish in the supermarket report a significantly lower incidence of fish poisoning attributable to scombroids.

We presume that an undetermined proportion of the poisonings reported are the result of some sort of bacterial decomposition. In many "mild" cases the only complaint is of gastrointestinal distress; the neurological symptoms specific to ciguatera and ciguatera-like toxins are not noted. Many of these cases may be attributable to scombroid poisoning but the symptoms may not be sufficiently pronounced for a proper diagnosis to be made.

The distinct histamine-like toxicity caused by bacterial degradation of the flesh of fish of the family Scombridae is a relatively common type of fish poisoning throughout the Caribbean. The symptomology includes a distinct "sharp" or "peppery" taste upon eating the fish followed by intense headache, dizziness, a variety of circulatory disfunctions, gastrointestinal distress, dryness of the mouth and inability to swallow. These symptoms are followed by generalized erythema, the face becomes swollen and flushed, eyes are sunken and an urticarial eruption may develop covering the entire body. In severe cases there may be additional complications of shock and respiratory distress. Death has been reported in a few cases but acute symptoms generally dissipate in 8 to 12 hours. This toxic reaction is brought about by the bacterial degradation of histidine in scombroid muscle tissue which produces a substance designated as scombrotoxin. Scombrotoxin probably has a combination of chemical constituents including saurine, histidine and possibly other toxic compounds. The disease responds well to treatment with anti-histaminic drugs; this specific treatment has mitigated the severity of scombroid poisoning as a public health problem in recent years.

In the eastern Caribbean scombroid poisoning has been reported from *Acanthocybium solandri* (wahoo), *Scomberomorus cavalla* (kingfish or king mackerel), *S. regalis* (spanish mackerel) and *S. maculatus* (cero). We are not aware of cases reported recently from the eastern Caribbean in the tunas *(Auxis, Euthynnus, Sarda, Scomber, Thunnus)* but these genera may also be incriminated. There probably is no true seasonality to scombroid poisoning although the incidence in any one area can be correlated with local "runs" of the particular species involved. Thus there seem to be more poisonings during the tourist season when sport fishing pressure is high.

The third general type of fish poisoning is described as ciguatera fish poisoning. Evidence from the Pacific suggests that there are at least three (probably more) distinct toxins capable of producing the ciguatera syndrome. Many authors (and many physicians in the eastern Caribbean area) have not separated the diagnosis or treatment of ciguatera from that of scombroid poisoning and some confusion has resulted. Both ciguatera and scombroid poisoning have been occasionally reported from the same fish in the Pacific (Halstead, 1967); we have no such reports from the Caribbean in recent years.

SYMPTOMOLOGY AND PUBLIC HEALTH ASPECTS OF CIGUATERA

In the absence of precise chemical and biogenic data we have defined ciguatera in terms of its symptomology. The following symptoms are extracted from Halstead (1967), Bagnis (1970) and Banner (1971) and are quoted as they appear in Teytaud and Brody (1971).

> Ciguatera fish poisoning in its simplest uncomplicated form develops within 3 to 5 hours after the fish is eaten. There is a sudden onset of abdominal pain followed by nausea, vomiting, and a watery diarrhea. The gastrointestinal symptoms will occur in about 40 to 75 percent of the cases. The victim feels weak, generally ill, and may experience muscle aches throughout the back and thighs in about 10 percent or more of the cases. Soon after, the victim complains of numbness and tingling in and about the mouth which then extends to the extremities (present in about 50 percent or more of the cases). Fever, headache, and rash are generally absent, and the patient has no desire for food. The acute symptoms usually subside in about 8 to 10 hours, and within 24 hours after onset most of the patient's symptoms will have completely subsided except for a feeling of weakness. However, the numbness and tingling may continue to a lesser extent for a period of 4 to 7 days. The foregoing resume is typical of the majority of uncomplicated ciguatoxications that are generally encountered by the practicing physician in an endemic ciguatoxic locality.
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> Ciguatera, like many other diseases, may vary greatly in its clinical manifestations depending upon the toxicity of the fish that is eaten, the individual's sensitivity to the poison, amount of fish ingested, and other factors. In a broader sense ciguatera fish poisoning may be characterized as follows: the onset of symptoms may vary from almost immediately to within a period of 30 hours after ingestion of the fish, but is usually within a period of 6 hours. The initial symptoms in some cases are gastrointestinal in nature, consisting of nausea, vomiting, watery diarrhea, metallic taste, abdominal cramps, and tenesmus, whereas in other patients the initial symptoms consist of tingling and numbness about the lips, tongue, and throat. This may be accompanied by a sensation of dryness of the mouth. The muscles of the mouth, cheeks, and jaws may become drawn and spastic with an accompanying sensation of numbness throughout. Generalized symptoms of headache, anxiety, malaise, prostration, dizziness, pallor, cyanosis, insomnia, chilly sensations, fever, profuse sweating, rapid weak pulse, weight loss, myalgia, and back and joint aches may be present in varying degrees, or one or more of these symptoms may be entirely absent. The victims usually complain of a feeling of profound exhaustion and weakness. The feeling of weakness may become progressively worse until the patient is unable to walk. Muscle pains are generally described as a dull, heavy ache, or cramping sensation, but on occasion may be sharp, shooting, and affect particularly the arms and legs. Victims complain of their teeth feeling loose and painful in their sockets. Visual disturbances consisting of blurring, temporary blindness, photophobia, and scotoma are common. Pupils are usually dilated and the reflexes diminished. Skin disorders are frequently reported that are usually initiated by an intense generalized pruritus, accompanied by erythema, and maculopapular eruptions, blisters, extensive areas of desquamation - particularly of the hands and feet - and occasionally ulceration. There may also be a loss of hair and nails.
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> In severe intoxications the neurotoxic components are especially pronounced. Paresthesias involve the extremities, and paradoxical sensory disturbances may be present in which the victim interprets cold as a "tingling, burning, dry-ice or electric-shock sensation", or hot objects may give a feeling of cold. In regard to the paradoxical sensory disturbance (P.S.D.), a classic example is that of a naval officer who was poisoned by an amberjack. Four weeks later he was observed subconsciously blowing on his ice cream, which was "burning his tongue", in order to cool it. Ataxis and generalized motor incoordination may become progressively worse. The reflexes may be diminished, muscular paralyses may develop, accompanied by clonic and tonic convulsions, muscular twitchings, tremors, dysphonia, dysphagia, coma, and death by respiratory paralysis. The limited morbidity statistics show a case fatality rate of about 12 percent. Death may occur within 10 minutes, but generally requires several days.

Table 1 summarizes the symptoms occurring during the first 24 hours after ingestion as they were reported by 25 persons who were interviewed following ciguatoxications of minor to moderate severity in St. Thomas, U.S.V.I. during 1971. Several of these reports represent the symptoms produced in different individuals by a single fish; they therefore do not represent 25 separate outbreaks.

TABLE 1

Summary of Symptoms Manifested by 25 Ciguatoxicated Individuals During First 24 Hours After Ingestion of Fish[1]

Symptom	Percentage Reporting
Abdominal pain	96 (96)
Nausea	88 (92)
Vomiting	68 (68)
Diarrhea	96 (96)
Numbness, tingling about mouth	56 (64)
Headache	48 (48)
Numbness in extremities	48 (56)
Metallic taste	24 (36)
Weakness	96 (96)
Muscle aches	40 (48)
Paradoxical sensory disturbance	32 (32)
Itching	64 (68)

[1]Percentages in parentheses represent change in original descriptions following questions by the interviewer.

Several additional symptoms were reported by three or fewer (less than 12%) of the persons interviewed. These symptoms include lack of coordination, muscle spasm, high fever, visual disturbances, diminished reflexes and skin rash. It is notable that none of these persons required hospitalization and only three reported visiting a physician (several others contacted a physician by telephone during the time period 24 - 72 hours after ingestion).

Virtually all of the persons interviewed reported noticeable symptoms of ciguatoxication for several days after the onset of the incident. Most commonly reported was extreme weakness and lethargy lasting up to 2 weeks. Many victims reported gastrointestinal symptoms well into the third day along with itching and/or skin rash. Those persons reporting the paradoxical sensory disturbance stated that it persisted for at least 3 days, in some cases 10 days or 2 weeks. The bulk of the other symptoms noted were reported as having dissipated within the first 24 hours.

We are currently undertaking a more extensive epidemiology reporting program in cooperation with local media, physicians and public health authorities. Data from this survey combining questionnaire and interview procedures should be available early in 1972. At this time we have no accurate estimate of the magnitude of the ciguatera poisoning problem in the Virgin Islands or for that matter anywhere else in the eastern Caribbean. Outbreaks in

Puerto Rico are sufficiently notable to receive coverage in the major English-language media which suggests that they are infrequent. The officials responsible for public health record-keeping in St. Thomas, on the other hand, estimate three or four cases per week are seen in the emergency room; if our 25 cases reported in Table 1 represent typical reporting ratios there may be as many as 30 cases per week in St. Thomas. These figures probably represent the maxima however and cannot be confirmed. Reports of ciguatoxication in the British Virgin Islands have stated that virtually "everybody" has been poisoned at least once (some as many as 15 times) but medical advice is almost never sought. Interestingly the British Virgin Islands are the only demographic unit mentioned by Halstead (1970) where fish poisoning is "not regarded as deterrent to the development of the fisheries programme".

The pattern of sporadic reporting of ciguatera poisoning despite the relatively high incidence of the disease is common throughout the northern Leeward Islands. Information from Halstead (1970) and our own contacts with fishermen, inter-island traders, charterboat operators and fishery personnel in the region suggests that the problem is indeed severe. Virtually every person contacted from St. Kitts, St. Maarten, St. Eustatius, Anguilla and Montserrat had either been a victim himself or knew of a close friend or relative who had been poisoned within the last 5 years. Very few of these cases were brought to the attention of a physician; most public health officials believe that "only the very severe cases are brought to the attention of the medical authorities" (Antigua: Halstead, 1970). One long-time resident of St. Kitts estimates only about one case in ten receives medical attention; as might be expected the bulk of the cases reported involve tourists and non-native residents.

GEOGRAPHIC DISTRIBUTION

The geographic distribution of ciguatoxic fish in the northern Virgin Islands is shown in Figure 1. The island of St. Croix, on a separate geological platform, is not reported as producing ciguatoxic fish in any appreciable quantity and has been omitted from this figure. A large number of the areas indicated have been reported as producing toxic fish for centuries. It cannot be presumed that these are the only localities; toxic fish are frequently caught in other areas.

In the Virgin Islands there is an extremely strong feeling among the fishermen that the south side of the Virgin Bank from Sail Rock east to Peter Island consistently produces toxic fish. Other fishermen would extend this area east and north to include most of the coastline of Virgin Gorda, some would include the Horseshoe Reef and Anegada. Still others (particularly those who regularly fish this southern Bank) state that only specific locations in this area produce toxic fish and that reef areas or "banks" only a few miles away are free of ciguatera. Virtually all fishermen feel that the entire north side (the Atlantic side) of the Bank is free of toxicity with the exception of a very few species. This pattern of geographic distribution of ciguatoxic fishes seems consistent with those areas reported by earlier authors (Walker, 1922; Arcisz, 1950; Brown, 1945; de Sylva, 1956; Mann, 1938). Other writers quoted in Halstead (1967), notably Hill (1868); Rogers (1899) and Gilman (1942), are contradicted by local fishermen, at least for the bulk of the species implicated elsewhere in the Virgin Islands.

In almost all reports on the geographic locality where toxic fish are caught the interviewee was referring to depths of 30 fathoms or less; the bulk of reports

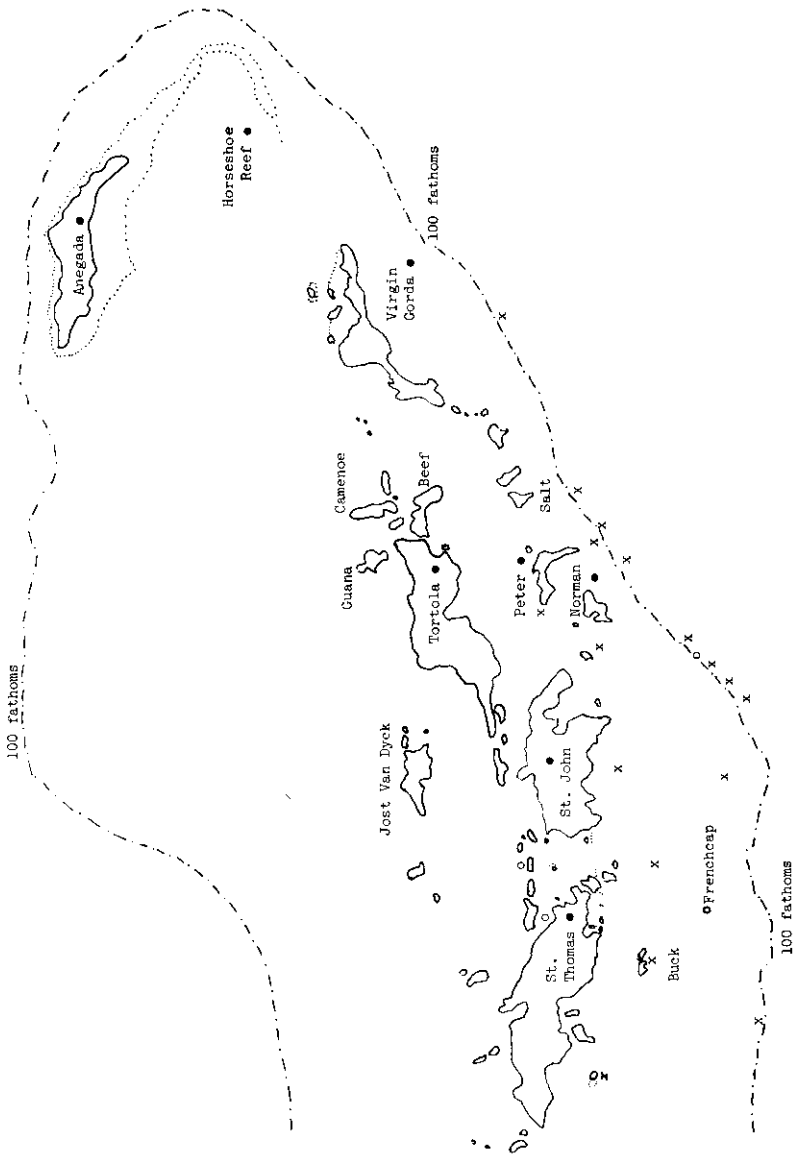

Fig. 1. Map of the Virgin Islands. • Islands or areas mentioned in the literature as ciguateric. X Sites of reported ciguatoxications 1968-1971. —·— 100 fathom contour. ····· shallow reef.

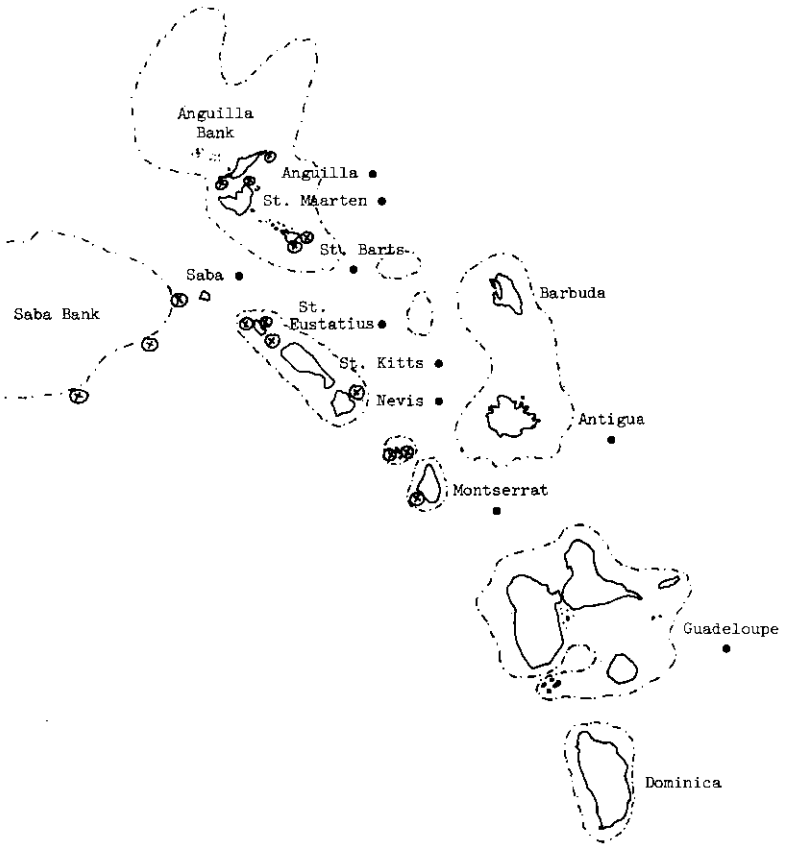

Fig. 2. Map of the Leeward Islands. • Islands reported in the literature as producing ciguatoxic fish. ⊗ Specific localities reported as toxic. —•— 100 fathom curve.

refer to reef areas in 8 - 15 fathoms, but this may be an artifact of fishing methods rather than a biological distribution. The relationship of depth to capture of ciguatoxic fishes is discussed in a later section.

The areas reported (by our contacts and by Halstead, 1970) as producing toxic fish in the northern Leeward Islands are shown in Figure 2. These data are plotted along with areas mentioned as toxic by various authors in Halstead (1967) and do not represent an intensive survey. More specific data will be presented in a later paper. The more southerly group of islands in the eastern Caribbean (Martinique south to Trinidad: The Windward Islands) have not commonly been reported as producing ciguatoxic fish in this century. Earlier authors make reference to a variety of species and locations but this is not confirmed by present residents.

Although no quantitative data are available it seems clear that the majority of ciguatera poisoning outbreaks in the eastern Caribbean occur in a rather small

area from Montserrat north to the Virgin Islands including all of the northern Leeward Islands and portions of Saba and Anguilla Banks. Beyond this area ciguatera poisoning is limited to sporadic outbreaks which generally involve large specimens of only a few species.

SPECIES REPORTED AS CIGUATOXIC

More than 400 species of fish have been implicated in ciguatera poisoning on a global basis (Bagnis, 1970). Of these 400, 91 species could conceivably be found in the eastern Caribbean. It is possible that an even larger number could be associated with ciguatoxications if they were desirable as food fish. There are also a number of reports of molluscs, crabs and lobsters producing the disease. Appendix I lists the 24 fish most frequently reported as toxic in the Virgin Islands. All of these species are valued as food fish with the exception of barracuda and amberjack; these two species have such a bad reputation as ciguateric that only the smallest specimens can be sold. It is somewhat surprising that the moray eel is as highly esteemed as it is, considering reports of toxicity from the Pacific. In St. Thomas eels are typically purchased by individuals from Spanish Caribbean cultures (Puerto Rico, Dominican Republic, Cuba), areas where ciguatera is less frequently reported. The fish at the top of the list tend to be reported as toxic more frequently than those lower down.

In general the larger specimens of these species are more frequently incriminated in ciguatera poisoning incidents. The fish generally can be considered "shore-fishes" or "reef-fishes" and for the most part conform to the pattern noted by other authors (Randall, 1958; Halstead, 1967; Banner, 1971): toxic fishes are not common at depths greater than 80 - 100 fathoms. Two notable exceptions in the Virgin Islands are the blackfin snapper, *Lutjanus buccanella,* and misty grouper, *Epinephalus mystacinus.* We have data on two outbreaks affecting five people from blackfin snapper caught in deep water and three additional outbreaks implicating misty grouper (which has not been taken at depths of less than 55 fathoms) involving at least ten persons during mid-1971. These data appear in Appendix II. Additional data on ciguatera from deep-living species is noted in a later section of this report. Although Banner (1971) states "true ciguateric fishes appear to be only those fishes tied directly to the flora and fauna of coral reefs. . . . ." there is excellent clinical data to support these outbreak reports; several members of our staff were among the victims. Samples have been retained for extraction and bioassay to quantify the toxicity of these fish (see also section on fisheries development).

CHEMISTRY AND PHARMACOLOGY

Our knowledge of ciguatera poisoning in the eastern Caribbean is presently based upon clinical reports and is only beginning to be quantified by chemical and biological assays. On the basis of symptomology and from the species implicated it is highly probable that ciguatera poisoning in the eastern Caribbean is produced by very similar (if not identical) compounds to those known from the Pacific. Scheuer and other workers at the University of Hawaii have isolated what they consider to be the primary toxin and, in cooperation with Hashimoto and his colleagues at the University of Tokyo, several secondary toxins. The primary toxin (deemed ciguatoxin) is insoluble in water, soluble in polar organic

solvents, heat stable to 100C, stable below 0C as a crude toxin but unstable in the semipurified or purified form unless extracted, purified and stored in an inert atmosphere at low temperatures. The non-crystalline product has the empirical formula $(C_{35}H_{65}NO_8)_n$ and the molecule has indications of a quaternary nitrogen atom, one or more hydroxyl groups and a carbonyl function. It is not a phospholipid. A crystalline product is currently undergoing analysis to determine its structural formula (Banner, 1971).

Present evidence (again from the University of Hawaii group) suggests that ciguatoxin acts upon excitable membranes to increase the permeability of Na + ions, upsetting the ionic balance of the membrane. Ciguatoxin is not an anticholinesterase *in vivo,* despite earlier papers, and various therapies for ciguatoxication based on stimulation of cholinesterase cannot be endorsed at the present time. The toxin is active at the level of 0.025 mg/kg when injected into mice with a toxin yield of 5 - 10 mg/kg from highly toxic flesh. The toxin is carried at a uniform level throughout the musculature of toxic fish but may be 50 to 100 times as concentrated in the viscera, particularly the liver.

Our laboratory in St. Thomas is currently using an acetone - diethyl ether extraction with purification by silicic acid column chromatography developed by Scheuer (pers. comm.) and bioassay using intraperitoneal injection into 20± gram Charles River CD-1 mice. We have previously used other extraction techniques including crude aqueous extraction with emulsifiers and a variety of experimental bioassay techniques. Our conclusions are basically the same as the workers in Hawaii although based on much less experience: careful solvent extraction and purification are necessary, rigidly controlled bioassay procedures are required and experienced laboratory personnel are an absolute requirement. To date there has been no rapid, simple colorimetric or other chemical test for ciguatoxin developed. Screening programs are exceedingly expensive and difficult to manage and are only in operation in Japan on a limited basis for selected samples from highly suspect areas. As much as we might desire it, we are not very close to a rapid means of identifying ciguatoxic fish in the laboratory and even further from a simple test which might be part of a housewife's shopping kit.

The traditional West Indian methods of determining if a fish is ciguatoxic have been discussed at length by previous authors. Appendix III lists these methods as reported to us by natives of the Virgin and Leeward Islands. Many housewives swear by some particular method utilizing visual inspection of external characters of the fish. Most admit that in practice both the visual methods and those requiring addition of some indicator are unreliable. We have submitted each of these methods to an assay with at least two known toxic fish and two non-toxic fish and have not found them reliable.

BIOGENESIS AND TRANSMISSION OF CIGUATOXIN

At the present time we have no accurate data on the mechanisms of biogenesis of ciguatoxin (or its related compounds) nor information on its transmission through the food chain. Banner, Helfrich, Randall and others at the University of Hawaii have concentrated a good deal of effort on these problems in the Pacific and their findings to date are summarized below (from Banner, 1971): (1) No causative agent or organism has yet been identified as producing toxins similar to ciguatoxin. (2) No definite evidence has been found to suggest that: (a) copper or other metallic ions act as chelators, trace minerals or catalysts

in the formation of the toxin; (b) no demonstrable increase in ciguateric fishes was noted in areas where "new surfaces" were exposed by natural disasters, dredging, blasting or predation by *Acanthaster;* and (c) contamination of the marine environment by pollutants (specifically wax esters at Wake Island) have no effect on ciguatera. (3) Normally non-toxic omnivores can be made toxic when fed small amounts of toxic fish over a period of time. (4) Toxic *Lutjanus bohar* retain toxicity for up to 30 months when fed a non-toxic diet. (5) A detrital feeding acanthurid *(Ctenochaetus striatus)* has demonstrable ciguatoxin in the flesh, viscera and gut contents. (6) Most carnivores seem to carry the same toxin (ciguatoxin) although additional toxins (eg: Aluterin, ciguaterin) may also be present. It should be noted that Dr. Banner will present a paper entitled "Biological Origin and Transmission of Ciguatoxin" tomorrow (18 November 1971), which could shed some new light on this subject.

Given the similar symptoms and species distribution reported in Pacific and eastern Caribbean ciguatera poisonings, it is reasonable to assume that similar biogenesis and transmission of the toxin can be expected. There are several persistent beliefs among eastern Caribbean fishermen which will be repeated here although we have been unable to confirm them.

1. Ciguatera is produced by fish which eat the fruit or leaves of manchineel (*Hippomane mancinella*). This theory has been proposed since 1511 (by Peter Martyr of Anghera; Halstead, 1967) and is probably based on advice to early explorers from Caribbean Indians. *Hippomane* is certainly toxic but its pharmacological action is quite different. It is doubtful that this theory is correct.

2. The most persistent theory in the Virgin and Leeward Islands involves copper. Natural copper deposits ("copper banks") are presumed to exist and fish which feed on these banks become toxic. Some of the more sophisticated fishermen suggest that it is not actually the copper metal but a small "sea moss" (which grows in areas where copper concentrations are high) which actually manufactures the toxin or a precursor. The "sea moss" responsible has been pointed out to us by several fishermen (actually three species: *Enteromorpha lingulata* from shallow water at Buck Island, St. Thomas; *Cladophora* sp. from fish pot warps south of Flanagan Island, U.S.V.I.; *Chaetomorpha* sp. from rocky subtidal at Buck Island, St. Thomas). None of these algae showed toxic activity when extracted with Tween and injected I.P. into mice; we plan to repeat this experiment with solvent - solvent extraction and column chromatography when these algae can be collected from historically toxic areas. Most fishermen suggest that the production of toxic "sea moss" is seasonal with peak growth in late spring or early summer. The association of ciguatoxin with copper is not limited to copper banks by fishermen; it is proposed that shipwrecks (particularly older wrecks with copper-sheathed bottoms) and copper antifouling paints supply all of the copper needed. We can in no way confirm the theory of copper-induced ciguatoxin at this time.

3. A theory proposed by a few fishermen and completely unproven at this time attempts to explain the high concentrations of toxic fish on the south of the Virgin Bank (with the lower toxicity reported from the same species on the north side of the Bank) and in the area from Antigua north to the Anegada Passage. It is proposed that the toxin is produced by some organism (presumably a primary producer) which is found only in areas where deep, cold, nutrient-laden water is upwelling. The theory is reasonable when applied to the southern Virgin Bank and the southeastern portions of Saba Bank, both noted as

producing toxic fish, as there is good evidence that upwelling does indeed occur in the Anegada Passage. The upwelling process cannot be confirmed in the St. Kitts – St. Eustatius – Redondo area at the present time due to lack of data. The specific organism(s) responsible and the mechanism of toxin production are not known by the proponents of this theory.

We are, therefore, no closer to an accurate description of the biogenesis or transmission mechanisms of ciguatoxin than purely theoretical considerations. We have proposed a series of studies similar to those undertaken by the University of Hawaii group including chemical, ecological and epidemiological programs for the next 3 years to attempt a better understanding of this problem.

EFFECTS OF CIGUATERA POISONING ON THE DEVELOPMENT OF FISHERIES

We have every reason to believe that ciguatera poisoning is a major impediment to the sale of local finfish in the Virgin Islands and thus is a strong deterrent to expansion of the commercial fisheries. Interviews by Halstead (1970) suggest that this is true throughout the Leeward Islands too, although residents of Antigua, St. Kitts and St. Maarten express the opinion that there is little alternative to continued buying of local fish and risk of intoxication.

In Dammann's 1967-68 survey of commercial fisheries of the Virgin Islands slightly more than half of total finfish consumption was from local (U.S. and British V.I.) sources (1,672,400 of 3,084,373). We have no data on how much of the fish imports could be replaced by local production if ciguatera were not a problem. Interviews with fishermen suggest that very few hotels and restaurants catering to the tourist trade would purchase locally caught grouper, snapper, jacks and kingfish because of fear of fish poisoning. Dammann's Table 9 "Fisherman-reported problems in the Virgin Islands commercial fishery" does not include any data on this subject, however Table 12 indicates that only two (2 of 79) of the fishermen contacted felt that there were "no fish" (commonly reported as ciguatoxic) so one might assume that ciguatera was indeed considered a problem.

Two investigations of fisheries development potential in the Virgin Islands area have recently been completed. The first (Dammann *et al* 1970) developed lines of approach carried out in the second project (Brownell and Rainey, 1971) for expanding the Virgin Islands fisheries through exploitation of deep water stocks. This effort was motivated by several natural limiting factors on the shallow water fish populations, among them the risk of ciguatera poisoning. It now appears that even species previously considered non-toxic because their normal depth-range is greater than 100 fathoms are implicated in ciguatoxications. Brownell and Rainey (1971) report three outbreaks from misty grouper (*Epinephelus mystacinus*) taken at 130-135 fathoms and the only documented case of ciguatera from a silk snapper (*Lutjanus vivanus*) from 110 fathoms. Two questionable outbreaks are reported by Dammann *et al.* (1970) from *Epinephelus nigritus* (actually *E. mystacinus*). In addition to the outbreaks reported for *E. mystacinus* and *Lutjanus buccanella* in Appendix II, we are aware of several outbreaks from *L. buccanella* attributed to fish caught during the exploratory fishing projects of the UN/FAO Caribbean Fisheries Development Programme (CFDP) in 1970 and '71. We are currently extracting and bioassaying about 2 tons of fish caught on UN/FAO cruises from areas where toxicity is reported. These data will be reported in early 1972. Although these data will

provide us with a more precise estimate of the proportion of ciguatoxic fishes in the deep shelf – shelf-slope populations, we have already ascertained that this resource is not free of ciguatoxin.

The toxic blackfin snappers caught by the CFDP came from Saba and Anguilla Banks; fishermen in Montserrat reported that most known poisonous fish had been captured in deep water — up to 250 fathoms (Halstead, 1970). It is highly probable that the abrupt dropoff to depths of 200 fathoms or more surrounding many of the Leeward Islands harbor excellent stocks of food fish but it is quite likely that some of these species carry ciguatera poisoning.

Halstead's 1970 survey found that fishermen, fisheries officers and public health officials were almost unanimous that ciguatera was a deterrent to development of the commercial fisheries. Most islands reported ciguatera in fish from depths of 0-60 fathoms and the most frequently toxic are all among the first ten species listed in Appendix I. At least two large commercial fishing operations in St. Maarten have given up shallow water fishing because of repeated ciguatoxications by their catch; several fishermen have reported having to discard large catches of jacks and grouper because their previous catches had caused poisoning. In the small communities of the Virgin and Leeward Islands an individual fisherman is occasionally completely boycotted because of his reputation for regularly landing toxic fish. Fishermen are expected to be able to determine whether or not a particular fish is toxic; an occasional exception is accepted, however.

The fisheries of the Virgin and Leeward Islands do not lend themselves to exploitation by large vessels with modern ground fishing gear. The pelagic stocks are apparently not sufficient to support a much larger fishing effort than is currently in progress. There are probably not sufficient stocks in the shelf-edge populations to withstand intensive fishing pressure equivalent to the Gulf of Mexico – Florida Straits snapper industry. The majority of fishermen in the eastern Caribbean are owner-operators of small boats (20 feet or less) who rarely go more than 10 miles from their home port. These fishermen could be trained and proper gear could be utilized for exploiting the area's natural stocks both in shallow and deep water but fish poisoning cases would be likely to increase. A thorough understanding of the ciguatera problem must be developed before expansion of the fishery can be effectively accomplished.

SUMMARY AND CONCLUSIONS

1. Fish poisoning in the eastern Caribbean is reported from all of the islands of the northern Virgin and Leeward Islands groups. Puerto Rico, Hispaniola and St. Croix have a much lower incidence rate as do the Windward Islands (Trinidad to Martinique).

2. Although clupeoid, elasmobranch, tetraodontoid and hallucinatory fish poisoning are reported from the eastern Caribbean, scombroid poisoning and ciguatera poisoning are considered to be most important. Because scombroid poisoning can be prevented by modern preservation techniques and treatment of the disease is specific and effective, it is considered a less severe problem than ciguatera poisoning.

3. Epidemiological reporting of ciguatoxications has only been begun in the last month throughout the Virgin Islands and a careful survey of the Leeward Islands must await additional funding. Ciguatera is presently reported as a severe public health problem with only a fraction of the cases reaching medical

attention. The problem seems most severe in the area from Montserrat north to the British Virgin Islands including the southeast portion of Saba Bank and the southern shelf of the Virgin Islands plateau.

4. The chemistry, pharmacology and ecology of ciguatoxin and closely allied compounds are at present poorly understood. The symptomology and species distribution of the toxins in the eastern Caribbean strongly suggest that a situation exists which is very similar to that described from the Pacific islands by the Marine Biotoxins group at the University of Hawaii over the past 16 years.

5. Toxicity in eastern Caribbean fishes seems to be more prevalent among the large carnivores of reef or reef-related habitats. There are a number of data which suggest that ciguatoxin(s) are produced by some organism in the reef food web and that the toxin is passed through the food web without significant modification and is concentrated by the larger carnivores.

6. Development of the commercial fisheries in the eastern Caribbean is severely impeded by the prevalence of ciguatoxin in commercially desirable species. There is good evidence that the shelf-edge stocks of snapper and grouper are not free from ciguatera poisoning as previously proposed and that exploitation of this presently underutilized resource may be impeded by this toxicity.

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APPENDIX I

Species most commonly reported as toxic in the Virgin Islands area

Species	Common Name
Sphyraena barracuda	Great barracuda; barra
Seriola dumerili	Amberjack; amber
Caranx latus	Horse-eye jack
Caranx ruber	Bar jack; carang
Caranx hippos	Crevalle jack
Lutjanus jocu	Dog snapper; dog tooth
Mycteroperca venenosa	Yellowfin grouper; gramminix
Scomberomorus cavalla	Kingfish; king mackerel
Caranx fusus	Blue runner; hard nose
Gymnothorax funebris	Green moray (probably Conger or Congo eel)
Epinephelus adscensionis	Rock hind
Mycteroperca bonaci	Black grouper
Scomberomorus regalis	Cero (often mistaken for "spanish mackerel")
Caranx lugubris	Black jack
Lutjanus griseus	Grey snapper
Lutjanus buccanella	Blackfin snapper
Epinephelus mystacinus	Misty grouper (often erroneously called Warsaw grouper)

Epinephelus guttatus	Red hind
Seriola rivoliana	Almaco jack
Caranx bartholomaei	Yellow jack
Apsilus dentatus	Black snapper
Epinephelus morio	Red grouper
Lachnolaimus maximus	Hogfish
Belistes vetula	Queen triggerfish; old wife

APPENDIX II

Outbreak reports from fishes caught at depths greater than 500 feet during 1970-71 (data from interviews; Ciguatera Case Repository numbers refer to CRI files)

CCR-71-011. Blackfin snapper - *Lutjanus buccanella* - about 4 lbs. Fish caught south of Frenchcap Cay, U.S.V.I. at about 40 fathoms by local fisherman. Fish was eaten by three people all of whom reported abdominal pain, loose bowels, nausea and vomiting, in that order. Onset between 3 and 6 hours after ingestion. Secondary symptoms included extreme weakness, listlessness and itching which developed 12-24 hours after ingestion and lasted for several (5 - 7 days). None of the victims reported previous intoxication.

CCR-71-018. Blackfin snapper - *Lutjanus buccanella* - about 3 lbs. Fish was purchased on the waterfront at St. Thomas by young couple visiting relatives on the island. Fish was broiled with sauce, no symptoms developed until about 8 hours after ingestion. Vomiting, diarrhea, weakness in the knees and dull headache persisted all the following day. Late afternoon produced P.S.D. for man but not wife. Returned to mainland 3 days after ingestion, no follow-up available.

CCR-71-008. Misty grouper - *Epinephelus mystacinus* - 36 lbs. Fish purchased at the dock - caught (apparently) south of St. John. Victims bought 5 lbs (two large steaks); refrigerated them and cooked fish next evening. Four persons had dinner of this fish; three young men and a young lady - one man and the lady reported nausea, vomiting and weakness within 6 hours; headache, nausea and weakness persisted for "3 or 4 days". The third victim had no violent symptoms of gastrointestinal origin but was lethargic and felt "weak in the joints" next day. The fourth person did not report any illness.

CCR-71-021. Misty grouper - *Epinephelus mystacinus* - 56 lbs. Fish caught by local sport-commercial fisherman at the "Warsaw Pocket" (misnamed since the area produces misty groupers) - about 3-1/2 miles south of Norman Island, B.V.I. at depth of about 120 fathoms. Fish was filleted and headed; at least six persons ate fillets with no ill effects. Two more people made soup of the head; they reported some intestinal discomfort and weakness, tingling sensations and lethargy the following day. Five other persons fried a small section of the liver: each reported eating "not more than a few bites" that night. All awoke within 3 hours with violent

abdominal cramps, vomiting and violent headache. Severity of gastro-intestinal symptoms continued for 6 hours or so, then extreme weakness, sinus-like headache and watery bowels persisted for 2 - 4 days. P.S.D. and tingling and numbness in the lips were reported about 16 hours after ingestion by three of the five. All reported persistent symptoms of weakness and soreness in all body joints for 7 - 10 days. P.S.D. persisted for at least a week in two victims.

CCR-71-023. Misty grouper - *Epinephelus mystacinus* - about 30 lbs. Steaks were sold to about four persons none of whom apparently developed ciguatera symptoms. A soup was made of the head and eaten by three persons. All described gastrointestinal distress, diarrhea and nausea within 3 - 6 hours; apparently the symptoms disappeared within about 24 hours for two of the victims; the third reported listlessness, weakness and achy joints which lessened by the third day after ingestion.

APPENDIX III

Methods for identifying ciguatoxic fish as described in the folklore of the Virgin and Leeward Islands (from interviews; Dammann *et al.*, 1969; Halstead, 1967)

I. External characters of the fish or fish flesh which indicate toxicity:

- More yellow or brassy color, especially about the head
- Stripes (in species where they are not normally obvious)
- Darker coloration
- Red coloration to the eyes
- Yellow mucus on inner lining of gullet
- Green tint to raw flesh
- Tiny black "veins" running through the musculature
- Brassy or coppery odor to the flesh
- Teeth are black
- Suspect species with roe is toxic
- Enlarged or bloated stomach
- Flesh tastes bitter or hot in mouth

II. Indicator organisms which suggest toxicity:

- Worms in the flesh (particularly jacks and mackerel) (Worms in the stomach indicate a non-toxic fish)
- Isopod ectoparasites ("cockroach") are not found on toxic fish (jacks)
- Flies will not land on flesh
- Ants will not eat

III. Methods employing an indicator:

- Silver turns black when boiled with toxic fish
- Sweet potato turns black when boiled with toxic fish